left side was removed by operation when it was found to involve the prepatellar bursa and to be adherent below to the patella.

Both microscopic (Fig. 3) and gross examinations showed a typical gumma.

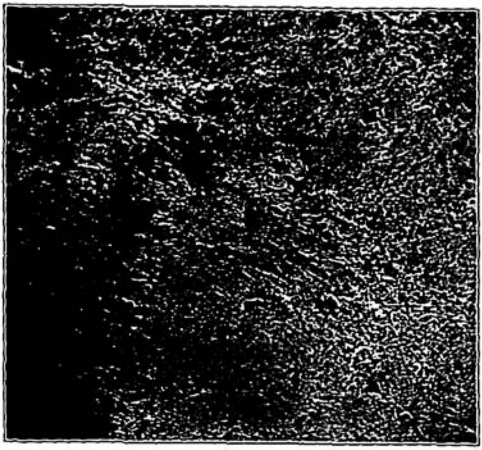

Fig. 3.

The patient was given antisyphilitic treatment for three months at the end of which time the remaining gumma was resolved.

Circumference of knees over midpatellar region now measured 30½ cm.

## A CONSIDERATION OF CARDIOSPASM, WITH THE REPORT OF A CASE.

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Cardiospasm, once thought to be a rare disease or condition, is being recognized much oftener on account of our increasing familiarity with its symptoms and the technical measures necessary to its direct diagnosis.

Only slightly more than a decade ago Mikulicz[1] estimated that about 100 cases had been reported. Since then Gottsteim[2] has added 25 additional cases. Bassler[3] speaks of 27 cases that he has treated. Plummer[4] has placed on record 40 cases. Besides these larger series of cases the following additional ones have been published during 1914 by Guisez,[5] Long,[6] Leuiller and Guisez,[7] and Reed,[8] and the subject has been treated by Geppert,[9] Heller,[10] Verger[11] and Eschbaum.[12] Geppert's article constitutes one of the best monographs on the subject.

The etiology of cardiospasm is a varied one. Many causes have been advanced and all are doubtless capable of proof in certain cases. The following causes have been suggested:

1. Primary cardiospasm (Meltzer).
2. Primary esophagitis (Martin).
3. Primary atony of the esophageal musculature (Rosenheim.)
4. Functional disturbance of the innervation of the esophagus, due to paralysis of the vagus causing simultaneous spasm and atony of the musculature of the esophagus (Kraus).
5. Congenital disposition (Fleiner, Zenker, Luschka and Sievers).
6. Kinking at the hiatus esophagei (Plummer).

Bassler[13] has recently published an article in which he puts forward the view that cases exhibiting obstruction at the lower end of the gullet of the type which we have hitherto thought to be cardiospasm is not cardiospasm but rather "a spasm of the esophageal opening of the diaphragm due to contraction of the muscular fibers of the crura . . . which contracts the esophageal opening by drawing the central tendon of the diaphragm against the front of the esophagus or contracts it at the sides."

In support of his view, which is based on a dissection of 5 fresh cadavera and Roentgen-ray observations of 7 cases of cardiospasm, he seems able to prove (1) that "the lower extremity of the esophagus or the cardiac orifice of the stomach have no or only a faintly developed sphincter," and (2) that the stricture is almost always epicardial, usually occurring at a distance of one vertebra above the cardiac orifice of the stomach, which corresponds to the esophageal opening of the diaphragm.

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[1] Quoted from H. S. Plummer, Collected Papers of St. Mary's Hospital, Mayo Clinic, Rochester, Minn., 1905-09.

[2] Keen's Surgery.

[3] Diseases of the Stomach and Upper Alimentary Tract, p. 829.

[4] Jour. Am. Med. Assn., August 15, 1908.

[5] Bull. Soc. de pédiat. de Paris, 1914, xvi, 144 to 150.

[6] Buffalo Med. Jour., 1913-14, lxix, 403 to 409.

[7] Jour. de méd. de Paris, 1914, 2 s., xxv, 413.

[8] Lancet-Clinic, Cincinnati, 1914, cxi, 378.

[9] Centralbl. f. d. Grenzgeb. d. Med. u. Chir., Jena, 1914, xviii, 149 to 187.

[10] Mitt. a. d. Grenzgeb. d. Med. u. Chir., Jena, 1913, xxvii, 141 to 149.

[11] Jour. de méd. de Bordeaux, 1914, xliv, 401.

[12] Med. Klin., Berlin, 1914, x, 141.

[13] New York State Jour. Med., New York, 1914, xiv, 9 to 11.

Bassler[14] offers no etiological factors tending to produce this diaphragmatic contraction of the esophageal opening. While his paper throws a new light upon the subject, it would appear to be one concerning proper nomenclature, affecting somewhat the pathology but not altering appreciably the symptomatology or therapy of what we now call cardiospasm.[15]

Under normal conditions we know that food on entering the esophagus requires from seven to ten seconds to pass into the stomach —about one-seventh to one-quarter of this time is required for food to pass down to the cardiac portion of the esophagus, where it remains for several seconds until the cardiac sphincter relaxes and the food is passed into the stomach by the peristaltic contraction of the circular and longitudinal fibers of the esophageal muscles. We have ample proof of this (1) in the Roentgen-ray studies of Kronecker and Meltzer, Cannon and Moser, and others, (2) by the common personal observation of experiencing the sudden aching pain of momentary duration felt back of the sternum in its lower third on swallowing too hot or too cold foods, such as soup, coffee, ice-cream, etc., and (3) by the postmortem evidence, after the ingestion of corrosive poisons, that the greatest amount of erosion in the esophagus occurs at its lower third in the neighborhood of the cardia, where contact with the corrosives has been longest sustained.

While there is this normal delay of foods at the cardia, this varies also with the character of the food, its temperature and chemical concentration, the extremes of all these causing a variable inhibition of the dilating mechanism of the cardia.

Idiopathic or primary cardiospasm occurs frequently, as is seen so often in hysteria and in those of neurotic temperament and tendencies, and is here purely functional; but if of frequent occurrence it permits more and more of the retention of foods, often irritating in character and composition, for too long a time in the neighborhood of the cardia predisposing to the development of esophagitis of varied types, erosions, fissures and ulcerations. Thus a vicious circle is produced, the local inflammation disposing to cardiospasm and the spasm permitting of unduly long retention of foods at the cardia, thereby increasing the esophagitis.

The commonest causes of cardiospasm are *primary cardiospasm* of a purely functional type and constituting a local neurosis, or a manifestation of a general neurosis, and *primary esophagitis*; but when both cardiospasm and esophagitis can be demonstrated simultaneously, it is extremely difficult to decide which is primary and which is secondary.

Of the other etiological factors, congenital disposition may be an active element in certain isolated cases. Indisputable proof of this, however, should be furnished by the history and the absence of all other etiological factors before one could feel reasonably safe in assigning this as the cause. Likewise, primary atony of the esophagus, while it may rarely occur, is probably also a less common factor in the etiology. Plummer, to whom we are largely indebted for our better understanding of this condition, concludes from a study of his cases that primary atony of the esophageal musculature is a rare occurrence, stating that in his cases "the almost invariable history of spasm at the outset, followed in the later period by the evidence of dilatation—that is, retention of food in the esophagus—is most convincing evidence that the spasm precedes the dilatation and that primary atony is rare." Functional disturbances of the innervation of the esophagus due to the paralysis of the vagus, and kinking at the hiatus esophagei, would also appear to be unusual etiological factors.

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[14] Loc. cit.

[15] Heed and Gross in an excellent review of the subject conclude that "cardiospasm is primarily the outcome of a hyperirritability of the vagus." Jour. Am. Med. Assn., January 22, 1916.

SYMPTOMS.—There are undoubtedly many cases of cardiospasm so slight as not to give rise to any subjective symptoms and in patients upon whom the diagnosis can be established only by mechanical means. The first subjective symptom usually volunteered by patients is a sensation of discomfort felt behind and usuallyto the left of the lower end of the sternum. This sensation is variously described as a dull, aching pain, sometimes throbbing, sometimes burning, sometimes cutting or a sense of pressure or weight, as if something had lodged low down in the gullet. These symptoms occur only during the ingestion of food and at first may be of short duration, with periodic remissions, during which the patient is able to eat freely and without dysphagia. During this period the esophageal musculature is sufficiently strong to overcome the spasm and permit of the entrance of food into the stomach with only momentary delay. As the condition progresses compensatory hypertrophy of the musculature must develop to overcome the increasing obstruction, and here a second symptom makes its appearance, namely, the regurgitation of foods from the esophagus into the mouth, very shortly after their ingestion, due to the overactive contracting efforts of the esophageal musculature. The regurgitated foods in this stage may be both liquid and solid, returning in practically the same condition as when eaten and not unpleasant in taste or odor. The majority of the food eaten is passed through the cardia slowly, but nevertheless surely, so long as the hypertrophied muscle proves itself competent to overcome the obstruction. Gradually the muscles tire under their added strain and rupture of the muscle bundles takes place with a resultant dilatation. At this stage the regurgitation of food may be temporarily less frequent and occur at somewhat longer intervals after food ingestion. As the dilatation becomes more extreme the esophageal capacity becomes greater and capable of retention of larger quantities of food, which are apt to be regurgitated only when the patient is lying down, stooping over, or during a paroxysm of coughing. The dilatation of the esophagus in time may become extreme, with a capacity well over a pint. Since the propulsive power of the esophageal muscle is lacking, food can reach the stomach slowly even when the spasm has been relaxed and in proportion to the weight of the column of food in the esophagus, assisted by gravity. The liquid portion of the meal usually passes more rapidly, seeping through the solid portion so as to leave a dense pultaceous, often foul-smelling mass, usually incorporated with tenacious mucus, which gives rise to a continual sensation of pressure and fulness behind the sternum, with occasional difficulty in breathing, due to pressure on the trachea. As food products are retained for longer and longer periods within the esophagus fermentation and decomposition, chemical and bacterial, take place resulting in secondary esophagitis. At this stage the condition of some patients is truly deplorable; they suffer continually with a sense of burning pressure back of the sternum; they are able to eat only small quantities of food at a time, and their total amount of food ingested and assimilated is so small that they lose weight rapidly; and if not relieved may develop a profound cachexia and die, literally of starvation.

DIAGNOSIS. The symptom-complex is usually so characteristic as to suggest the diagnosis, although I must confess that it is not always so easy as it sounds, for in the case here reported I was at first inclined to believe it one of bronchiectasis. The direct diagnosis can always be made by the use of an esophageal bougie, preferably of the Plummer type, very often by means of the stomach-tube or by fleuroscopic study and the Roentgen-ray plate analysis. In the early stages, with the use of the esophageal bougie or of the stomach-tube, it will be found that an obstruction to the further passage of the instrument in adult cases is met with at about sixteen or seventeen inches from the incisor teeth. The instrument can be passed readily until this point is reached, when an elastic-like obstruction is met with, which under firm and steady pressure usually gives way and permits of the passage of the bougie or the tube into the stomach. During the periodical remissions the obstruction will not be met with, or at times the bougie or tube seems about the pass when it is suddenly gripped in the spasm of the contracting muscle which has been excited by the instrumentation. Great care should be practised in attempting to pass the instrument beyond the obstruction, until the diagnosis of cardiospasm is definitely made to the exclusion of diverticuli, a kinking at the hiatus, stenosis due to malignant disease from within or without the esophagus, or from external pressure by an aneurysmal sac or mediastinal tumor.

To rule out these differential possibilities the use of the Roentgen-ray diagnosis had best be first employed, which will disclose the presence of a thoracic aneurysm, mediastinal growth, the presence of diverticuli, and the irregular outlines of a carcinoma infiltrating the lower end of the esophagus and causing a stenosis. The use of Bassler's cardioplugger[16] will be of aid in securing better Roentgen-ray plates of esophageal conditions. After aneurysm and mediastinal tumor have been eliminated it is safe to proceed with further instrumentation. If either a diverticulum, a kinking of the hiatus esophagei, or a carcinomatous stenosis is suggested by the Roentgen-ray examination it is best to make use of the esophageal bougie devised by Plummer, which consists of a series of olive tips, which are attached to a stout whalebone staff. The olives are perforated from about their middle to the tip. Six yards of thread are then swallowed by the patient, preferably three yards one afternoon and three yards the following morning, which permits of the thread passing well down into the upper coils of the intestines and becoming fixed so firmly that strong traction can be made on the proximal end emerging from the mouth. The olive tip is then passed over this thread, and by means of traction on the thread the olive tip can be safely guided through the cardia. By varying the amount of traction the sound can be introduced into a diverticulum and its depth and size determined.

The diagnosis of cardiospasm may likewise be confirmed by a test which I believe has hitherto not been reported—namely, by esophageal lavage. With the tip of a stomach-tube in the esophagus at a point just above the obstruction, water is allowed to run in from a graduated glass tank. It will be seen to run much more slowly than if the tube were in the stomach. From 100 to 500 c.c., according to the amount of esophageal dilatation, will run in slowly, but evenly until the flow suddenly stops and the level of fluid in the graduated glass tank begins to oscillate slightly. At this point the water is allowed to escape through the outflow tube, and without changing the position of the stomach-tube it will be seen that the amount recovered is equal to the amount introduced. When the capacity of the esophagus has been reached, if instead of opening the outflow tube the fluid is allowed to remain in the esophagus, by its weight, assisted by gravity, it will cause the cardiospasm to relax. This will occur in a varying number of seconds, according to the degree of the spasm, and will permit some of the water in the esophagus to pass into the stomach, thereby allowing an additional flow from the glass tank, which will then usually proceed in a uniform way until recovered by introducing the tube *farther into the stomach.* While for purposes of demonstration this latter step may be permissible, it is never wise to distend the esophagus to its point of capacity, as it exaggerates the already existing atony. The essential point in diagnosis by this method is the ability to recover *from the esophagus itself* an amount of fluid equal, or nearly so, to that introduced. This is not possible of accomplishment in diverticulitis or in stenosis or partial stenosis of the cardia due to pressure from within or without. *It is also of prime importance to determine the presence of a secondary or complicating esophagitis.* This is possible by means of the esophagoscope, but its use is so formidable to the patient that I prefer to make the diagnosis by the examination of esophageal sediments obtained by a method recently published.[17] The photomicrographs used in connection with this paper were made from sediments obtained by this method. It is necessary to determine the extent and kind of this complicating esophagitis because it indicates the proper application of the principles of treatment, *for as long as the inflammatory condition exists so long will the cardiospasm persist,* notwithstanding efforts directed toward the latter to the neglect of the former.

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[16] Bassler, Diseases of the Stomach and Upper Alimentary Tract, p. 829.

TREATMENT. In the earlier cases of cardiospasm of the primary type, relief may usually be obtained by the administration of antispasmodics, such as belladonna and atropin, pushed to the limit of tolerance; and with due regard to a possible neurotic etiological factor, the regulation of proper hygiene, and the use of hydrotherapy and exercise, preferably in the open air, should be advocated. If these measures do not suffice, esophageal bougies may be used or the spastic cardiac ring may be dilated by means of dilators of the types suggested by Plummer and by Bassler. When there is a concomitant esophagitis, measures should be adopted toward allaying this before proceeding to, or simultaneous with, the treatment of the cardiospasm. Suitable measures are the lavaging of the esophagus with medicated solutions best determined and controlled by the character of the esophageal sediment If the esophageal erosion or ulceration has become secondarily invaded by bacteria one can use germicidal solutions, such as potassium permanganate, silver nitrate, argyrol, etc., until the bacteria have disappeared from the inflammatory desquamation, when blander solutions, such as boric acid or normal salt solution, can be substituted. The use of an autogenous vaccine prepared from cultures grown from the esophageal sediments will facilitate recovery from the severer types of esophagitis. If the esophagitis is sharply localized in the form of ulcerations or erosions, healing medicaments may be directly applied by means of long applicators introduced through a small-bore esophagoscope or through a rubber tube just long enough to reach from the incisor teeth to the lower end of the esophagus. When the condition has progressed to the stage of esophageal dilatation and atony the use of intra-esophageal electricity is indicated, preferably with the sinusoidal current or the faradic current by means of a suitable intragastric electrode. The negative pole should be attached to the electrode within the esophagus and the positive pole to the external electrode in the form of a hand sponge, which is to be carried over the transverse processes from the seventh cervical to the third dorsal vertebræ and over the sternomastoid muscles, particularly the left, to stimulate the vagus. Before turning on the current the patient should drink a small glassful of water to serve as a better conductor of electricity and to prevent burning by direct contact. The duration of each treatment should not exceed ten minutes, and should be given daily in severe cases until improvement is noted. In those cases showing progressive loss of weight due to inanition it is important to arrange the diet in the form of liquids, the total caloric value of which for twenty-four hours should be over 3000 calories. This can be accomplished by the liberal use of milk and cream, olive oil, butter, egg-nog, soft-boiled or raw eggs, and non-stimulating broths.

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[17] Lyon, AMER. JOUR. MED. SCI., September, 1915, No. 3, cl. 402.

In the very late cases that come under observation during the extreme stage of starvation weakness it is perhaps better to do a preliminary gastrostomy and feed directly through the stomach and try to build up the strength of the patient before proceeding to the other treatments. Apropos of operative procedure, Bassler[13] suggests that "in intractable cases the approach to the site and cause of the stricture had best be made by the safer abdominal route rather than through the thorax, and that an operation which has to do with the division of the crura, either at their insertions or the bisection of two of the inner portions of both at the back of the gullet or some point in the esophageal opening is worthy of consideration."

It is necessary to observe certain patients over a long period of time. Relapses from primary cardiospasm, properly treated, are comparatively uncommon, but relapses due to exacerbations in any residual esophagitis are more frequent. This will be seen in an examination of Chart II.

PROGNOSIS. Properly treated patients do well, except possibly those instances of cardiospasm secondary to an esophagitis caused by the action of corrosive poison, which are much more difficult to heal and result in contractions due to scar tissue, which, though healed, may still predispose to spasm. It was my privilege to see 2 such cases in Berlin in Paul Cohnheim's clinic which had been treated respectively for five and nine years.

The following case is reported because it illustrates how stubbornly certain cases may persist when the causative factor is an esophagitis, how relapses are frequent and are due to an exacerbation in the esophagitis and that it is necessary that this should be allayed before the vicious circle of cardiospasm and esophagitis can be broken.

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[13] Loc. cit.

CASE REPORT.—M. J., aged thirty-two years; occupation, piano stringer. Born in Russia.

*Chief Complaint.* The patient first came under observation December 18, 1912, with complaint of "stomach trouble," epigastric pain, morning cough, copious expectoration, and loss of weight.

*Family History.* Negative.

*Past Medical History.* Always well and strong until three years ago, and the only illness he remembers was typhoid fever at the age of eighteen years.

*Social History.* The patient came to America seven years ago, has been married five years, and has two children in good health. Does not use tobacco. Uses light wine occasionally. Denies venereal disease.

*Present Illness.* Beginning about two years ago the patient noticed an uncomfortable feeling of fulness in the high epigastrium with a sense of weight and pressure occurring shortly after his meals, preceded by a slight stabbing pain referred to just above the xiphoid, which would occur irregularly and be associated with the first few mouthfuls of food taken, and would soon pass off to be replaced by a feeling of weight and heaviness. There was no nausea or vomiting but considerable belching, with partial relief and occasionally sour regurgitations. The patient has been steadily losing weight and strength during the past three years. Seven years ago he weighed 205 pounds, and today weighs 144 pounds (subsequently further loss of weight to 128 pounds), and the greater part of this has been lost during the past year.) Two or three months ago he began to be troubled with coughing, and would sometimes spit up a mouthful of very thick, rather foul-smelling sputum, which would seem to suddenly well up in the back of his throat. This had gradually become worse until now, particularly when lying down, and especially when getting out of bed in the morning, he has paroxysmal coughing, which ends with the spitting up of large quantities of thick, tenacious bad-smelling sputum. Of late he has been having night sweats. On some mornings he may spit up as much as a pint and a half of sputum.

*Physical Examination.* Frail, anemic-looking adult male; looks tired out and ill; skin dry and cold.

Eyes negative; no pupillary alteration. Hair, nose, and mouth are negative. Teeth are in good condition. Gums and conjunctivæ are pale. Glands negative. Breath is slightly offensive. Neck negative. *Chest:* long, narrow thorax, with flaring lower ribs. Supraclavicular fossa increased in depth, but equally. *Lungs:* Tactile fremitus increased at right apex and extends to third rib. The percussion note is dull over this area and particularly at the right posterior apex, where expiration is prolonged, and occasionally accompanied by rales. No sign of cavity formation. Heart is normal in size and position; second aortic sound slightly accentuated; no murmurs; muscular quality deficient.

*Abdomen.* Scars from furunculosis; soft; no distention; no rigidity; no masses; some tenderness to deep palpation one inch below the xiphoid; enlarged area of gastric tympany. Liver, spleen, kidneys, appendix, hernial orifices, genitalia are all negative. Deep reflexes sluggish. Pulse 84, rather low volume and tension; regular in rate; no sclerosis of peripheral bloodvessels. Respiratory rate 20. Temperature 99. Blood-pressure 110-80.

*Technical Examinations.* A stomach-tube passed three hours after a breakfast of scrambled eggs, bread and tea, a cup of coffee, and one glass of water recovered 500 c.c., a dense, tenacious, sour-smelling mass, taking all night to filter to 85 c.c. of turbid filtrate, testing as follows: Free HCl, 20; combined HCl, 25; total acidity 50. Occult blood negative.

In withdrawing the stomach-tube a paroxysm of coughing was induced and was followed by the spitting up of about 200 c.c. of very foul-smelling, purulent-looking material, grayish-white in color and exceedingly tenacious. This material was negative for free HCl.

December 19, 1912. Full dinner last night at 10.30. Now eleven-hour fasting stomach. Stomach-tube shows marked retention of many meat fibers, potato remains, spinach and prune skins. After lavaging the stomach, which required 3000 c.c. to clean, the stomach-tube was slowly withdrawn seven inches, and almost immediately from the end of the tube came about 400 c.c. of foul-smelling, dense tenacious material so mucoid and cohesive as to form a perfect rope, corresponding in size to the lumen of the stomach-tube and which coiled up snake-like in the conical glass receptacle. This material which is identical in appearance with the "sputum," microscopically contains only food rests, desquamated epithelial cells, many leukocytes, and large numbers of bacteria, chiefly streptococci. It is slightly acid in reaction to litmus, but negative for free or combined HCl. This finding, of course, essentially limited the source of this material to the esophagus, but did not differentiate esophageal diverticulitis, stricture, new growth, or cardiospasm. Subsequently it was noticed that at times the stomach-tube, No. 32 French, would pass easily and at other times with considerable difficulty, meeting resistance at a point about 40 cm. from the teeth. The patient was sent to the German Hospital, January 28, 1913, for further study and treatment and remained until June 13, 1913, under the service of Dr. Henry F. Page, to whom I am indebted for the privilege of studying and assisting in the treatment of this patient.

During the time the patient remained in the hospital his pulse-rate varied from 50 to 88. His respiratory-rate varied from 18 to 24 and his temperature from 96° to 100°.

The *sputum* was examined eighteen times for tubercle bacilli with negative results.

*Blood counts* showed a moderate secondary anemia with hemoglobin 77 per cent. on his lowest reading. There was a moderate *leukocytosis reaching 15,850, with normal differential* counts. Sputum inoculated into guinea-pigs, subcutaneously and intraperitoneally, as well as into the liver, did not noticeably impair

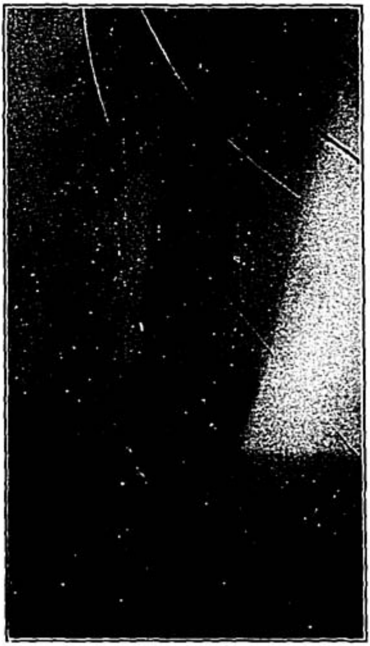


FIG. 1.—The plate from which this photograph was made was unfortunately broken, and it does not show the extent of the esophageal dilatation, which reached up to the first rib. (February, 1913).

the health of the animals which were macroscopically normal when killed and microscopic examination of tissues showed no evidence of tuberculosis or other infection.

*Roentgen-ray examination* of lungs shows a very small area at the extreme apex of right lung in which there is marked increase in density. The remainder of both lungs is apparently negative.

*Roentgen-ray plate and fleuroscopic examination* of stomach made by Dr. A. G. Miller, shows moderate peristalsis; the stomach is moderately dilated, the lower border being just below the umbilicus with the patient standing. The bismuth passes slowly into the stomach in a thin but steady stream, but the majority of it is retained in the esophagus and reaches a point level with the first rib, and the width of the esophagus measures from 1.8 to

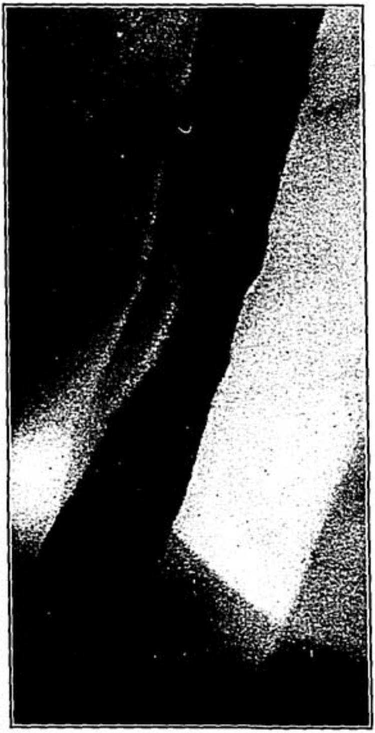
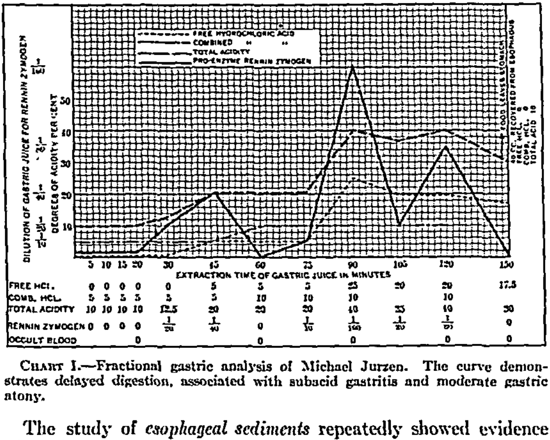


FIG. 2.—Shows the recovery of esophageal tonus. Note the excellent peristaltic wave. (October, 1915.)

4.3 cm., at different levels, being narrowest at the point where the esophagus reaches the diaphragm and widest at a point 18 cm. (7 inches) above the diaphragm (Fig. 1). (*Cf.* with radiographs made twenty-three months later, by Dr. Willis F. Manges, Fig. 2.) There is no evidence of diverticulitis or new growth within or without the esophagus. The Wassermann and Widal reactions were both negative. Esophagoscopy gave unsatisfactory results, nothing further being learned. The patient stood the operation well without the use of anesthesia. Fractional analysis of gastric contents, shows delayed digestion, subacid gastritis and gastric atony. (See Chart I.) Stool examinations were negative.



| | 5 | 10 | 15 | 20 | 30 | 45 | 60 | 75 | 90 | 105 | 120 | 150 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| FREE HCL. | 0 | 0 | 0 | 0 | 0 | 5 | 5 | 5 | 25 | 20 | 20 | 17.5 |
| COMB. HCL. | 5 | 5 | 5 | 5 | 5 | 5 | 10 | 10 | 10 | | 10 | |
| TOTAL ACIDITY | 10 | 10 | 10 | 10 | 12.5 | 20 | 20 | 20 | 40 | 35 | 40 | 30 |
| RENNIN ZYMOGEN | 0 | 0 | 0 | 0 | $\frac{1}{20}$ | $\frac{1}{40}$ | 0 | $\frac{1}{10}$ | $\frac{1}{160}$ | $\frac{1}{20}$ | $\frac{1}{80}$ | 0 |
| OCCULT BLOOD | | | | 0 | | | 0 | | 0 | | 0 | 0 |

CHART I.—Fractional gastric analysis of Michael Jurzen. The curve demonstrates delayed digestion, associated with subacid gastritis and moderate gastric atony.

The study of *esophageal sediments* repeatedly showed evidence of esophagitis, with marked desquamation of esophageal epithelium, often fragments of epithelium containing from eight to twenty layers of stratified squamous epithelial cells. Many slides examined showed evidence of ulceration, with areas of necrotic débris with large numbers of pus cells; many streptococci and occasional red-blood corpuscles and all manner of food rests. (See Figs. 3 and 4.) These sediment pictures continued during the first few months of treatment with a gradual subsidence in the inflammatory elements, and at present (August, 1915) the esophageal sediments are negative except for occasional exfoliation of stratified epithelial cells. There is too no microscopic evidence of food rests that were such a constant finding during the early months of observation.

The treatment practised in this case was as follows: (1) lavage

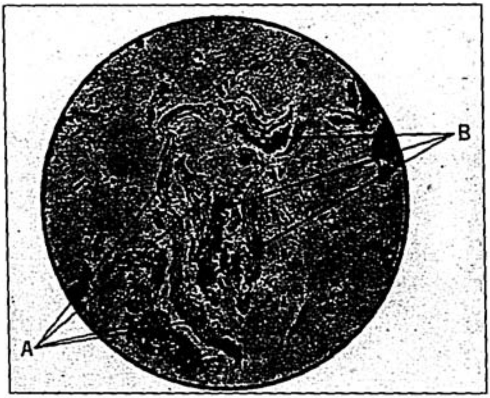


FIG. 3.—Esophageal sediment from a case of cardiospasm. M. J., aged thirty-two years, showing *A*, exfoliated stratified squamous epithelium from esophageal mucosa, with *B*, leukocytic infiltration. × —100.

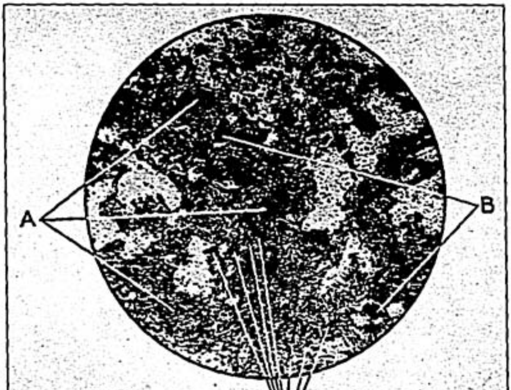


FIG. 4.—Esophageal sediment from a case of cardiospasm, with esophagitis. M. J., aged thirty-two years, showing *A*, exfoliated epithelium containing *B*, masses of streptococci, and *C*, leukocytic infiltration. × —100.

of the stomach with normal saline or various stomachics, such as quassia, condurango and gentian. After which the tube was withdrawn about seven inches, so that its tip rested in the cardiac portion of the esophagus and the esophageal residual contents were recovered either by simple expression or by being gently drawn off by a syringe. For several weeks the amount recovered each day varied between 200 and 500 c.c., and was very thick, tenacious, and very foul-smelling. (See Chart II.) The esophagus was then lavaged, at first using a solution of potassium permanganate in strength of 1 to 16,000 and 1 to 12,000, until the putrefactive process was under control, and later with solutions of normal salt. During this process of lavaging the esophagus it was found that all of the lavaging fluid could be recovered from the esophagus without having to pass the tube into the stomach. This, I believe, to be pathognomonic of cardiospasm. After lavaging the esophagus, olive-tip bougies, well lubricated, were passed by steady pressure through the spastic cardia, retained in place for several seconds, and then passed

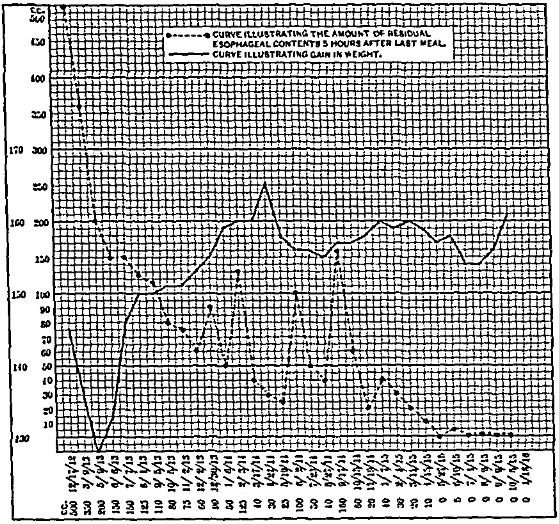


CHART II.—The curves illustrate the decrease in esophageal retention and the increase in body weight as the patient (M. J.) improved.

rapidly up and down from four to ten times. At first the tonicity of the spasm was so great as to permit the passage of olive tips, only 5 mm. in diameter, gradually increasing the size to tips 18 mm. in diameter, which can now be passed without effort.

After this an intragastric electrode, properly protected by a perforated hard-rubber tip and a capillary rubber tube, was passed into the esophagus, after the patient had swallowed a glass of water, and the faradic current, and subsequently the sinusoidal current, was used for periods of from five to ten minutes at each treatment. The negative pole being within the esophagus and the positive pole being attached to a small sponge electrode, which was carried over the sternocleidomastoid muscle on either side to stimulate the vagi and over the spinal cord between the seventh cervical and the third dorsal vertebræ. The frequency of treatment was gauged by the amount of the esophageal retention and its character, and by the amount of esophagitis, which was estimated by a study of the esophageal sediments made at frequent intervals. In the first few weeks the amount recovered each day varied between 200 and 500 c.c., and in character was very foul-smelling, and consisted of macroscopic and microscopic food rests, often fermenting, and held tenaciously together by quantities of thick glairy mucus, in which was found microscopically various food rests and many pus cells; stratified squamous epithelial cells from the esophagus, many bacteria, and an occasional red-blood cell. As improvement took place the amount of esophageal retention gradually lessened, and in six weeks had fallen below 150 c.c. Six months later the retention had been reduced to 50 c.c., and gradually became less and less until two months ago (about twenty-two months after treatment was begun), it became impossible to demonstrate esophageal retention of either macroscopic or microscopic food rests.

The *diet* was arranged on a plan of liquid foods of various forms—milk, cream, broths, oatmeal gruel, barley-water, albumen-water, egg-nogs, and olive oil—and was given in small amounts and at hourly intervals, so that the total caloric value for twenty-four hours varied from 3000 to 3500 calories.

Under this plan the patient gained in weight 16 pounds (from 128 to 144) during a period of two months. Gradually his diet was increased to soft and semisolid foods, the latter being gauged by the type of food rests found in the esophageal sediment. His weight is at present 165 pounds, which shows a net gain of 37 pounds. He is on full diet, has no symptoms, and expresses himself as being in better health than he can ever remember, and has been back at his work for about two years.